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A CASE OF EOSINOPHILE ASCITES: WITH REMARKS.

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EOSINOPHILE ascites has, so far as I can find out, hardly ever been recorded. This rather contrasts with the attention which has been devoted to the occurrence of eosinophile cells in the exudate of experimental acute peritonitis in its earliest stages, especially by Durham, Opie, Dudgeon and Ross. But the exudate of acute peritonitis in human beings rarely contains as much as 1 per cent. of eosinophile cells. In this case of eosinophile ascites acute peritonitis brought the eosinophile count down from 22 to 0.5 per cent. The explanation of a haemic eosinophilia of 12 per cent. is not obvious in the absence of animal parasites. Possibly it may be connected with the presence of carcinoma, for Weinberg and U. Mello [8] found 3.33 per cent. of eosinophiles in carcinoma of the colon, and 6.6 in carcinoma of the liver.

History and Course.

A male, aged 52, at the end of September, 1913, had colic and vomiting two days after eating some fish, followed gradually by jaundice and putty-like stools, and about the middle of October by transient pruritus. He was admitted to St. George's Hospital on November 4th with considerable jaundice, some rather vague resistance in the right loin, an enlarged and tender liver, and tenderness near the umbilicus. The gall bladder and the spleen could not be felt. For a time there was some apparent improvement, but on November 21st ascites was obvious, and on December 3rd 7 pints of fluid were removed; Dr. E. L. Hunt found that this contained red blood corpuscles, lymphocytes 46 per cent., endothelial cells 29, eosinophiles 22, polymorphonuclears 2, mast cells 1. The Wassermann reaction in the ascitic fluid and in the blood was negative (Mr. H. W. Bayly). A differential count of the haemic leucocytes (total count 17,040) showed: Polymorphonuclears, 71 per cent.; lymphocytes, 16.5; mononuclears, 0.5; eosinophiles, 12; mast cells, 0. Examination of the stools did not reveal the presence of any ova of worms. The urine constantly contained bile pigment, but not bile salts, and there was no sugar; in the last week of life there was a trace of albumin on two occasions. Early in December the base of the right lung was found to be dull on percussion, breath sounds to be absent, and skiagraphy showed opacity. On December 12th the patient had severe abdominal pain, and the pulse rose to over 100. The pain gradually increased and necessitated the use of morphine. On December

14th the patient became comatose and died on December 15th at 2 a.m. His temperature, which had always been about normal, rose to 99.6° F. on December 14th.

Necropsy.

The necropsy was performed by Dr. R. S. Trevor. The skin was universally jaundiced. There was acute peritonitis with a copious effusion which, Dr. E. L. Hunt reported, contained polymorphonuclear cells 79 per cent., lymphocytes and endothelial cells 21 per cent.; a count of 400 white cells showing two eosinophiles. There were numerous minute secondary growths on the peritoneum, especially on the diaphragm, which was much thickened. There was a primary "colloid" carcinoma in the ascending colon just above the ileo-caecal valve, which was not narrowed. The caecum was high up in the abdomen on the right side. The liver, 5½ lb., contained numerous secondary growths, some of which were "colloid." There was much matting in the portal fissure, but the portal vein was free from thrombosis. The walls of the gall bladder were white, thickened, and microscopically infiltrated with carcinoma. There were no biliary calculi. The kidneys, 8 oz. each, were bile stained, but otherwise normal. The spleen, 8 oz., was soft. There was a bilateral bile-stained pleural effusion; there was recent pleurisy on the right side; the fluid was not examined. There was extensive collapse of the lower lobes of both lungs. The apex of the right lung was free from tuberculosis, but near the apex of the lower lobe there was a small calcareous mass. There was no evidence of tuberculosis in the left lung, or of metastases in the lungs or pleurae. The heart, 11 oz., was soft, and the endocardium bile-stained. On the anterior cusp of the mitral valve there was a small serous cyst, the size of a sixpence; as this burst on examining the heart its contents could not be investigated.

Microscopically the cyst was shown to be due to oedematous separation between the superficial and the deep layers of the valve segment, and not to present any evidence of a hydatid cyst. There was no trace of hydatid disease elsewhere in the body. Microscopically the growth in the colon was a columnar celled carcinoma showing extensive myxomatous degeneration. Sections of the primary growth and of the peritoneal metastases did not show eosinophilia.

In this case the discovery of ascitic eosinophilia suggested hydatid infection of the peritoneum, on the ground that haemic eosinophilia may accompany hydatid disease. But I was somewhat surprised at the apparent absence of any published evidence that rupture of a hydatid cyst into the peritoneum caused a local eosinophilia in that serous sac. I therefore wrote for information to Professor F. Dévé, of Rouen, the authority on hydatid disease, who very kindly replied:

> Peritoneal eosinophilia does, indeed, sometimes occur as the result of intraperitoneal rupture of a hydatid cyst —with or without choleperitoneum—but, so far as I know, one case only has been recorded—namely, in Brouqueyre's thesis, "De l'éosinophilia générale et locale dans les kystes hydatiques" (*Thèse de Bordeaux*, 1905, p. 70). This observation was also given *in extenso* (without any fresh cytological details) in Tremblin's thesis, "De l'ouverture spontanée des kystes hydatiques du foie dans le péritoine" (*Thèse de Paris*, 1906, No. 283, p. 45, Obs. I). This is the extract from Brouqueyre's thesis: "M. Lereboullet has told me of an extremely interesting case which he watched when under the care

of Professor Gilbert. A hydatid cyst of the liver ruptured, but none of its contents passed into the peritoneum. The usual phenomena due to a secondary ascites of no great size led to paracentesis. The fluid withdrawn was deeply bile-stained, and, on centrifuging, gave a deposit containing a considerable quantity of eosinophile cells, at least 50 per cent.

I have seen a case of the same kind, which, though I intend to publish it eventually, you are at liberty to quote. In February, 1913, I diagnosed a hydatid of the liver in a man of 23 years of age, and advised operation without delay. Four days later it ruptured spontaneously when he was sitting quietly at his desk doing accounts. Laparotomy twenty-seven hours after the rupture revealed rupture of a hydatid cyst on the anterior surface of the left lobe of the liver and 3 or 4 litres of turbid fibrinous fluid the colour of orangeade and containing scolices. Bacteriological cultures proved that the fluid was sterile, and chemical analysis that it contained urea (0.45 per cent.), fibrin, cholesterin, bile pigments, and traces of glucose, but no bile salts. Cytologically I found polymorphonuclear neutrophiles 85 per cent., eosinophiles 8 per cent., small and medium-sized mononuclears 0.5, large mononuclears (macrophages) 0.5, and red blood cells 5.3 per cent. At the time of the operation the blood contained 2.7 per cent. of eosinophiles (before the rupture it was only 0.9 per cent.).

Dr. d'Este Emery has told me that on one occasion he found 10 per cent. of eosinophiles in the ascitic fluid of a man who afterwards died of abdominal malignant disease. I have not obtained any other information from published or personal sources about eosinophile ascites, but a few words may be added about eosinophile pleural and cerebrospinal effusions.

Eosinophile pleural effusions have been reported by Widal and Revaut[9] (4 cases), Barjon and Cade[1] (5 cases), Perkins and Dudgeon.[7] According to Emerson,[3] there is a slight eosinophilia (2 to 5 per cent.) in tuberculous pleurisy, and Mosny, Dumont, and Saint-Girons[4] state that eosinophile pleural effusions are common in Paris. In a traumatic haemothorax the latter observers found 92 per cent. eosinophiles, while the haemic eosinophile count was 4 per cent.; and in 2 cases of pleural effusion due to bullet wounds H. S. Carter[2] reported eosinophilia of 70.2 and 87.8 per cent. respectively. Eosinophile pleural effusions have been regarded as usually transient, benign, and as indicating a process on the way to cure (Widal and Revaut). Perkins and Dudgeon's case, however, proved fatal. Eosinophilia may occur in pleural effusions due to such widely different causes (post-pneumonic, post-typhoid, haemorrhage, granular kidney, rheumatism, tuberculosis) that its presence cannot be correlated with any special etiological factor. In many of the cases the effusions have been sterile on culture. With eosinophile pleural effusions there is usually, though not invariably, haemic eosinophilia, which, however, never approaches the count seen in the serous effusions. Mosny, Dumont, and Saint-

Girons contend that in these cases the eosinophilia is a local process, and that under some unknown influence the granulations of the polymorphonuclear neutrophile leucocytes become eosinophile, the curves of the number of neutrophile and eosinophile leucocytes in these effusions varying inversely. This hypothesis as to the origin of eosinophile cells is opposed to the usually accepted view, originally put forward by Ehrlich and subsequently supported by Opie,[6] that the eosinophile cells are always derived from the bone marrow.

Mosny and Harvier [5] report a case of chronic syphilitic *meningitis* in which four lumbar punctures were performed. In the first puncture fluid eosinophiles were absent, in the second they formed 0.3 of the cells present, in the third 5 per cent., and in the fourth 9 per cent. All transitions from lymphocytes to eosinophile cells were seen. The eosinophile cells in the blood were never more than 1 per cent. This was regarded as an example of local eosinophilia due to the transformation of lymphocytes into eosinophile cells, as stated by Dominici.

REFERENCES.

[1] Barjon et Cade, *Arch. gén. de méd.*, Paris, 1903, cxcii, 1859. [2] Carter, H. S., *Med. News*, New York, 1904, lxxxv, 629. [3] Emerson, C. P., *Clinical Diagnosis*, p. 621, 1906. [4] Mosny, Dumont, et Saint-Girons, *Arch. de méd. expér. et d'anat. path.*, Paris, 1912, xxiv, 488. [5] Mosny et Harvier, ibid., 1907, xix, 373. [6] Opie, *Amer. Journ. Med. Sci.*, Philadelphia, 1904, cxxvii, 217. [7] Perkins and Dudgeon, *Trans. Path. Soc.*, London, 1907. lviii, 119. [8] Weinberg et Mello, *Bull. Soc. path. exot.*, 1908, i, 463, [9] Widal et Revaut, *Compt. rend. Soc. Biol.*, Paris, 1900, 11 S., ii, 648.